

Demographic Recruitment in Sexually Transmitted Disease Models

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ABSTRACT

Models of STD dynamics customarily comprise a series of stages through which individuals may pass (susceptible, infected, infectious, recovered or dead, for example). Unless these models try to incorporate some aspects of age-structure (so that there are birth rates), the population considered is effectively that of sexually active adults, into which individuals are recruited at some given rate g . In the case of AIDS models, for example, g is almost invariably taken to be a constant; the implication is that the population to be modelled is part of a larger meta-population, and that the effects of infection within the population do not influence the demographic processes in the meta-population. It is of interest, however, to consider what happens if potential recruits from the meta-population are influenced by the size and disease status of the population as a function of the knowledge of disease prevalence--as in the case of AIDS or gonorrhea--that may be available to potential recruits. This knowledge may influence who enters the population as well as who returns to the meta-population. For example, fewer recruits may decide to enter the population when prevalence becomes large, or susceptibles or recovered may return to the meta-population (effects on internal recruitment). We approximate these behavioral demographic effects by making g a function of the population variables: g may be a decreasing function of the total number of infecteds, or of the fraction of the population infected. In this paper we will consider some of the consequences for STD dynamics when recruitment behavior based on prevalence occurs, using very simple models for populations. The models and results discussed in this manuscript represent a very crude attempt to incorporate demographic environmental effects that result in response to the current state of the epidemic embedded in a larger meta-population.

Keywords: risk behavior; sexually-transmitted disease; autonomous response; non-linear incidence, HIV/AIDS.

1. INTRODUCTION

There is some evidence that the perceived state of a biological system may indeed have a significant effect on the dynamics of a particular system. Changes in sexual behavior in response to information about the possibility of acquiring sexually transmitted diseases (STDs) may have a significant effect in determining the level of incidence or rate of spread of STDs particularly in this era of HIV/AIDS. Large changes of behavior have been confirmed in various homosexual subpopulations in San Francisco (McKusick *et al.* 1985a; Winkelstein Jr. *et al.* 1988; CDC MMWR 1985; Shilts 1987). These changes include the reduction in the number of steady partners from a median of 16 to a median of 1 (Third International Conference on AIDS, Washington D. C. 1987), a 40% reduction on the number of male partners per month and a 50% reduction on the frequency of anal intercourse without a condom (see the AIDS Behavioral Research Project as reported in McKusick *et al.* 1985b). These changes have taken place among homosexually active men in several regions including New York City (see Martin 1986, 1987) and Boston (see Saltzman *et al.* 1987).

Changes in behavior are capable of affecting the *recruitment* of new susceptibles (see Fineberg 1988 and references therein), the level of sexual *activity* (or average number of partnerships per unit time; see McKusick *et al.* 1985b), the type of sexual practices (see Baldwin and Baldwin 1988; Evans *et al.* 1989; Shechter *et al.* 1988), or the *mixing* of individuals (such as the systematic avoidance of individuals perceived to have a high probability of being infected; see Fox 1987, McCusker *et al.* 1988; van Griensven *et al.* 1989a, b; Wiktor *et al.* 1990; Curran *et al.* 1988 and Fineberg 1988). Changes in recruitment can be internal and external and may be due to various reasons. Individuals in the meta-population may choose not to enter it because of behavioral changes that are external to the explicit population under consideration, while individuals within the population may decide to leave it by drastically changing their behavior.

In the next sections, we study the external and internal effects of disease prevalence on recruitment in the context of the core group which consists of the most sexually-active members of the population at large (see Hethcote and Yorke, 1984). Here we only model explicitly the core population. Hence, exiting it does not mean the total avoidance of sexual activity, but rather a lack of participation in the most sexually-active network. Hethcote and Yorke (1984) found that the core group was the group responsible for generating the most cases of gonorrhea and that control measures directed at this group was the most effective way of reducing the level of endemicity--modelled by a "moving endemic equilibrium." Their results were based on a model that assumed that all the populations involved--including the core group--had constant population size.

In this manuscript, we assume that the core-population has variable population size and observe that in some situations behavioral changes in the meta-population (external recruitment) or in the core group (internal recruitment) will have the effect of reducing the size of the core group. Furthermore,

we provide an example in which drastic changes in behavior will substantially reduce the size of the core-group at equilibrium and may even destabilize it. In the latter case, we observe periodic solutions within the core population.

2. DEMOGRAPHIC RECRUITMENT MODELS

We consider a population--a core group--of total size N , and a sexually-transmitted disease. At any given time t , $S(t)$ members of the population are susceptible to the infection, and $I(t)$ members have been infected by the disease, $N = S + I$. The population dynamics are described by the model

$$\begin{aligned}\frac{dS}{dt} &= g(S,I) - \mu S - c \frac{SI}{N} \\ \frac{dI}{dt} &= c \frac{SI}{N} - (\sigma + \mu)I\end{aligned}\tag{1}$$

In this model μ denotes the per capita death rate in each class from causes other than the disease, c denotes the number of contacts sufficient to transmit the disease per infective in unit time, and σ denotes the per capita rate of departure from the infective class either through death from the disease, through recovery, and/or through transition to a class outside the population being considered. For the moment we assume that μ , c , and σ are positive constants, but we will consider generalizations later.

The term $g(S,I)$ represents the rate at which new members are added to the susceptible class, and we consider initially two quite different situations. In the first, $g(S,I)$ represents a birth rate while in the second it represents the rate of in- or out-flow of individuals from a larger meta-population. The first interpretation of $g(S,I)$ falls within the realm of classical epidemic models and a succinct discussion of potential applications follows.

Because our model does not include a removed or recovered class, we are essentially restricted in this case to a universally fatal disease--although we could allow recovery without immunity by adding a term aI ($a \leq \sigma$, internal recruitment) to $g(S,I)$. There are many universally fatal diseases for which (1) is a plausible model. In diseases such as brucellosis, where infectives contribute to the birth rate (Pugliese, 1990a, b) it would be appropriate to assume that the birth rate is a function of total population size

$$g(S,I) = G_1(S + I)\tag{2}$$

so that rate of change of g with respect to S and I are equal. Specifically, if we denote the partial derivatives of g with respect to S and I by g_S and g_I respectively, then $g_S = g_I$ and the birth rate has a purely demographic effect. For debilitating diseases in which infectives do not contribute to the birth rate, such as rabies (Anderson, Jackson, May and Smith 1981), it would be more appropriate to assume

that the birth rate is a function of the susceptible population size

$$g(S,I) = G_2(S) \quad (3)$$

so that $g_I(S,I) = 0$. In this case the birth rate has a demographic component and also an indirect epidemiological component because of the disease-induced reduction in the birth rate. It is reasonable to assume that $g(0,0) = 0$ and that the population has a carrying capacity K in the absence of diseases, so that

$$g(K,0) = \mu K, \quad g_S(K,0) < \mu. \quad (4)$$

In particular, (4) implies that the natural death rate μ is small enough so that $g(S,0) - \mu S > 0$ on some interval. We allow for the possibility of an Allee effect in the population dynamics, where the function $g(S,0) - \mu S$ is negative on some interval $0 < S < \alpha$ with $\alpha < K$ and $g(S,0) - \mu S$ is positive for $\alpha < S < K$. As the slope of the population growth rate function $g(S,0) - \mu S$ at $S = 0$ is $g_S(0,0) - \mu$, there exists an Allee effect if and only if

$$g_S(0,0) < \mu. \quad (5)$$

If there is an Allee effect,

$$g(\alpha,0) = \mu\alpha, \quad g_S(\alpha,0) > \mu. \quad (6)$$

If (5) is not satisfied we assume that $g(S,0) - \mu S > 0$ for $0 < S < K$, and in any case we assume

$$g(S,0) - \mu S < 0 \text{ for } S > K. \quad (7)$$

Ordinarily, the function $g(S,0)$ increases to a maximum and then decreases as S increases, so that $g_S(S,0) < 0$ for S sufficiently close to K . In particular for the special cases (2) and (3) we will assume $G'_1(N) < 0$ and $G'_2(N) < 0$ near K (the prime denotes differentiation). In general, for birth models one may assume

$$g_I(S,I) \leq g_S(S,I)$$

when $g_S(S,I) \geq 0$ and $g_I(S,I) \leq 0$ when $g_S(S,I) \leq 0$ to include the examples (2) and (3) and to allow for epidemiological effects in the birth process. It is also reasonable to require $g_S(S,I) \leq 0$ ($S \geq K$).

The main thrust of this manuscript will concentrate on state-dependent recruitment for sexually-transmitted diseases. We consider a population of sexually-active individuals whose behavior puts them at risk of infection. The underlying assumption is that the population explicitly modelled is part of a larger meta-population and that the effects of the infection within the population do not influence the demographic processes in the meta-population. These assumptions are not very limited; in fact, it is strongly believed that sexually-transmitted diseases are sustained by very small subpopulations. Hethcote and Yorke (1984) introduced the idea of core groups of individuals (sexually-active and very-sexually-active) to model the transmission dynamics and control of gonorrhea. It was implicitly assumed that both of these populations of sexually-active individuals were key to the survival of

gonorrhea in the set of all sexually-active individuals in the USA (the meta-population).

For fatal diseases such as HIV/AIDS the term σI in (1) represents a transition from the infected (HIV positive) class to the class of those with AIDS symptoms who are assumed to be sexually inactive. For diseases such as gonorrhea from which individuals recover without immunity, the term σI may represent recovery and return to the susceptible class. In this manuscript, we do not deal with the return of recovered individuals to the susceptible class (but see Section V), an extension that can be carried out by adding a term to the first Equation in (1) and following the approach outlined in this manuscript.

In models for HIV/AIDS (see Castillo-Chavez *et al.* 1989a, b, c, d, Thieme and Castillo-Chavez 1989, 1992, Blythe and Anderson 1988a, b, Jacquez *et al.* 1988, and references therein), the recruitment is taken to be constant, ruling out the possibility that potential recruits to the population may be influenced by the real or perceived prevalence of the disease in the population. The incorporation of the effects of disease prevalence in recruitment leads to the study of model (1) with $g(S, I)$ interpreted as the *net* recruitment rate into the susceptible class. We are allowing for both the entrance into and the exit from the susceptible population. Several assumptions can be made on the specific dependence of g on S and I . A natural assumption is that the recruitment rate depends only on the size of the infective class I and that it is a decreasing function of I . Explicitly, we have that

$$g(S, I) = G_3(I) \text{ and } g_I(S, I) = G'_3(I) \leq 0. \quad (8)$$

Another possibility is that the recruitment rate is a decreasing function of the per-susceptible incidence rate or of the proportion of the population which is infected, so that

$$g(S, I) = G_4\left(\frac{I}{S+I}\right) \text{ with } g_I(S, I) = \frac{S}{(S+I)^2} G'_4\left(\frac{I}{S+I}\right) \leq 0. \quad (9)$$

In general, if $g(S, I)$ represents a net recruitment rate from the meta-population, departure of susceptibles from the population is impossible if there are no susceptibles, and therefore we must assume

$$g(0, I) \geq 0. \quad (10)$$

For recruitment models, including the special cases (8) and (9) we will assume that (10) holds, and also that

$$g_I(S, I) \leq 0. \quad (11)$$

In order to consider the possible return from the infective class into the susceptible class (as is the case in gonorrhea) we would have to weaken condition (11). For (8) the recruitment effect is purely epidemiological while for (9) it has both demographic and epidemiological components.

For recruitment models dealing with the disease dynamics of populations embedded in a large

meta-population, it is not reasonable to assume that $g(0,0) = 0$, and we will assume $g(0,0) = \Lambda > 0$. Strictly speaking, some modification of (9) is needed for small total population sizes as $g(S,I)$ does not have a limit as $(S,I) \rightarrow (0,0)$ in this case. Mathematically the case $g(0,0) = 0$ can be handled but this technical exercise does not add any significant insights to this manuscript. The assumptions (4) and (7) are plausible for recruitment models as well as for birth models with $K = G_3(0)/\mu$ for (8) and $K = G_4(0)/\mu$ for (9). Thus we shall assume that (4) and (7) hold for recruitment models. While an Allee effect is possible even if $g(0,0) > 0$, we will not deal with this case further.

We will analyze the behavior of model (1) covering only the recruitment case and will cover the situations illustrated by examples (8) and (9). There are recruitment models in which the recruitment rate can be negative in some regions of the S-I plane: for example, one may envision a situation in which the real or perceived level of disease in a population is so high that individuals leave the population, abandoning their risky behavior before getting infected. Some of these situations are also covered implicitly in the analysis provided.

3. EXISTENCE AND STABILITY OF SOLUTIONS

In the absence of infection, $I = 0$, we have $S = N$ and the population size is described by the model

$$\frac{dN}{dt} = g(N,0) - \mu N \quad (12)$$

Since $g(0,0) > 0$, (12) has only the equilibrium $N=K$, which is asymptotically stable with domain of attraction $N > 0$. The two-dimensional model (1) has an equilibrium $(K, 0)$. In addition, there may be endemic equilibria (S_∞, I_∞) with $I_\infty > 0$. An endemic equilibrium is a solution with $0 < S < K$, $I > 0$ of the pair of equations

$$\frac{g(S,I)}{S} = \mu + \frac{cI}{N} = \mu + \frac{cI}{S+I} \quad (13)$$

$$cS = (\sigma + \mu)N = (\sigma + \mu)I + (\sigma + \mu)S.$$

The second of these equations gives

$$S = \frac{\sigma + \mu}{c - (\sigma + \mu)} I, \quad (14)$$

and thus an endemic equilibrium is possible only if

$$c > \sigma + \mu. \quad (15)$$

From (14) we deduce

$$S + I = \left[\frac{\sigma + \mu}{c - (\sigma + \mu)} + 1 \right] I = \frac{c}{c - (\sigma + \mu)} I, \quad (16)$$

and then substitution into the first condition of (13) gives

$$\frac{g(S, I)}{S} = c - \sigma.$$

Thus, by (15) at an endemic equilibrium (S_∞, I_∞) we have

$$\frac{g(S_\infty, I_\infty)}{S_\infty} = c - \sigma > \mu.$$

Because of (11),

$$g(S_\infty, 0) \geq g(S_\infty, I_\infty) > \mu S_\infty$$

and then $S_\infty \leq K$ because of (7).

According to (15), c cannot be too small. On the other hand, c cannot be too large in order to assure $S_\infty > 0$. For example, if $g_S(0, 0) > c - \sigma$ and $g_{SS}(S, 0) \leq 0$ for $0 \leq S \leq K$, it is not possible to have $S_\infty > 0$, and thus there is no endemic equilibrium. If we think of c as a parameter, we consider the value $c = \sigma + \mu$ as the value where an endemic equilibrium appears and when c becomes large enough, $c = \sigma + g_S(0, 0)$, the endemic equilibrium passes through $S = 0$. The condition (15) is necessary but not sufficient for the existence of an endemic equilibrium. For an equilibrium (S_∞, I_∞) with $S_\infty > 0$, the linearization of (1) has coefficient matrix

$$\begin{bmatrix} g_S(S_\infty, I_\infty) - \mu - \frac{cI_\infty}{N_\infty} + \frac{cS_\infty I_\infty}{N_\infty^2} & g_S(S_\infty, I_\infty) - \frac{cS_\infty}{N_\infty} + \frac{cS_\infty I_\infty}{N_\infty^2} \\ \frac{cI_\infty}{N_\infty} - \frac{cS_\infty I_\infty}{N_\infty^2} & \frac{cS_\infty}{N_\infty} - \frac{cS_\infty I_\infty}{N_\infty^2} - (\sigma + \mu) \end{bmatrix}$$

or

$$\begin{bmatrix} g_S(S_\infty, I_\infty) - \mu - \frac{cI_\infty^2}{N_\infty^2} & g_S(S_\infty, I_\infty) - \frac{cS_\infty^2}{N_\infty^2} \\ \frac{cI_\infty^2}{N_\infty^2} & \frac{cS_\infty^2}{N_\infty^2} - (\sigma + \mu) \end{bmatrix}.$$

At the equilibrium $(K, 0)$ the matrix is

$$\begin{bmatrix} g_S(K, 0) - \mu & g_I(K, 0) - c \\ 0 & c - \sigma - \mu \end{bmatrix}.$$

Because of (4), the eigenvalues of this matrix are negative if and only if $c < \sigma + \mu$. Thus by (15), the equilibrium $(K, 0)$ is asymptotically stable if and only if the basic reproductive or contact number

$$R_o = \frac{c}{\sigma + \mu} \quad (16)$$

is less than one and no endemic equilibrium is possible. If the contact number exceeds one, then $(K, 0)$ is a saddle point.

There can be an endemic equilibrium (S_∞, I_∞) only if the contact number exceeds 1, in which case

$$\frac{S_\infty}{N_\infty} = \frac{1}{R_o} = \frac{\sigma + \mu}{c}, \quad \frac{I_\infty}{N_\infty} = (1 - \frac{1}{R_o}),$$

where $N_\infty = S_\infty + I_\infty$ is the total population size at the endemic equilibrium. The coefficient matrix of the linearization takes the form

$$\begin{bmatrix} g_S(S_\infty, I_\infty) - c(1 - \frac{1}{R_o})^2 - \mu & g_I(S_\infty, I_\infty) - \frac{c}{R_o^2} \\ c(1 - \frac{1}{R_o^2})^2 & \frac{c}{R_o^2} - \mu - \sigma \end{bmatrix}.$$

However, the computations are most easily done if we replace R_o by $(c/(\sigma + \mu))$ everywhere in the above matrix including S_∞ and I_∞ . Using the original parameters, we see that the trace of this matrix is

$$g_S(S_\infty, I_\infty) - (c - \sigma)$$

and the determinant of the matrix is

$$\left(\frac{1}{c}\right) (\sigma + \mu - c) [(\sigma + \mu) \{g_S(S_\infty, I_\infty) - (c - \sigma)\} - (\sigma + \mu - c)g_I(S_\infty, I_\infty)].$$

As $\sigma + \mu - c < 0$ by (15), the conditions for the asymptotic stability of an endemic equilibrium -- that the trace of the coefficient matrix be negative and that the determinant of this matrix be positive -- are

$$g_S(S_\infty, I_\infty) < (c - \sigma) \quad (17)$$

$$(\sigma + \mu)\{g_S(S_\infty, I_\infty) - (c - \sigma)\} - (\sigma + \mu - c)g_I(S_\infty, I_\infty) < 0. \quad (18)$$

Since (11) is satisfied, (17) implies (18) and thus the condition (17) by itself is necessary and sufficient for asymptotic stability.

4. STABILITY OF ENDEMIC EQUILIBRIA

For recruitment models, in view of assumption (11), the condition for asymptotic stability of an endemic equilibrium is (17). Endemic equilibria are given by the intersections of the line through the origin

$$I = \frac{c - \sigma + \mu}{(\sigma + \mu)} S \quad (20)$$

and the curve

$$g(S, I) = (c - \sigma) S . \quad (21)$$

The curve (21) passes through a point $(S_0, 0)$ given by $g(S_0, 0) = (c - \sigma) S_0 > \mu S_0$, and by (7), this implies that $S_0 \leq K$. In order to guarantee the intersection of (20) and (21) and hence the existence of an endemic equilibrium we need some additional condition which will guarantee $I \rightarrow \infty$. For example, if (8) is satisfied the curve (21) is given by $(c - \sigma) S = G_3(I)$ and there is a point on the curve for $0 \leq I < \infty$. If (9) is satisfied, the curve (21) is given by

$$(c - \sigma) S = G_4\left(\frac{I}{S + I}\right) .$$

On any vertical line, $G_4\left(\frac{I}{S + I}\right)$ decreases from $G_4(0)$ when $I = 0$ to $G_4(1)$ as $I \rightarrow \infty$. If

$$S > \frac{G_4(1)}{(c - \sigma)},$$

there is a point on the vertical line where the curve (21) crosses the line, and

$$S \rightarrow \frac{G_4(1)}{(c - \sigma)}, \quad I \rightarrow \infty .$$

In fact, it is not difficult to see that for (8) and (9) the endemic equilibrium is unique because of the assumptions that the functions G_3 and G_4 are decreasing.

As the slope of the curve (21) is given by

$$\frac{dI}{dS} = \frac{g_S(S, I) - (c - \sigma)}{-g_I(S, I)} ,$$

an endemic equilibrium is asymptotically stable if and only if this slope is negative at a crossing of the

curve (21) and the line (20). In the special case (8) of a recruitment rate which depends on the number of infected members $g_S(S, I) \equiv 0$, the unique endemic equilibrium is asymptotically stable.

For a recruitment model with recruitment rate depending on the fraction of the population which is infected or in the incidence per susceptible as in (9), we let

$$\eta \equiv \frac{I}{S+I},$$

so that $\eta = 0$ corresponds to $I=0$ and $\eta=1$ corresponds to $S = 0$. At an endemic equilibrium,

$$1 - \eta = \frac{1}{R_o}, \quad \eta = (1 - \frac{1}{R_o}), \quad (22)$$

because of (14). This endemic equilibrium (S_∞, I_∞) is found by first calculating η from (22) and then solving $G_4(\eta) = (c - \sigma) S$ for S . Thus the equilibrium is asymptotically stable if and only if

$$g_S(S_\infty, I_\infty) = -\frac{I_\infty}{(S_\infty + I_\infty)^2} G'_4(\eta) < c - \sigma,$$

and using (22), we see that the endemic equilibrium is asymptotically stable if and only if

$$-G'_4(\eta) < (c - \sigma) \left(\frac{I_\infty}{(S_\infty + I_\infty)^2} \right) - 1 = \left(\frac{c - \sigma}{\eta} \right) \left(\frac{c}{\sigma + \mu} \right) S_\infty = \frac{G_4(\eta)}{\eta(1 - \eta)} \quad (23)$$

There are various possibilities for the stability of the endemic equilibrium as a function of η depending on the choice of the function G_4 . Consider for example

$$G_4(\eta) = \Lambda e^{-b\eta}$$

corresponding to the recruitment function

$$g(S, I) = \Lambda e^{-\frac{bI}{S+I}}.$$

Then $G'_4(\eta) = -bG_4(\eta)$ and the stability condition (23) is satisfied if

$$b < \frac{1}{\eta(1 - \eta)}, \quad 0 < \eta < 1.$$

As the function $\frac{1}{\eta(1 - \eta)}$ has a minimum value 4 on $0 < \eta < 1$, attained when $\eta = \frac{1}{2}$, the endemic equilibrium (S_∞, I_∞) given by

$$S_\infty = \frac{\Lambda}{c - \sigma} e^{-b\left(1 - \frac{1}{R_o}\right)}$$

$$I_{\infty} = \frac{(c - \mu - \sigma)\Lambda}{(\mu + \sigma)(c - \sigma)} e^{-b\left(1 - \frac{1}{R_0}\right)}$$

is asymptotically stable for every η , $0 < \eta < 1$, or every $R_0 > 1$, if $b < 4$. Thus the endemic equilibrium of the model

$$\frac{dS}{dt} = \Lambda e^{-\frac{I}{(S+I)}} - \mu S - c \frac{SI}{S+I}$$

$$\frac{dI}{dt} = c \frac{SI}{S+I} - (\sigma + \mu)I$$

($b=1$) is asymptotically stable whenever $R_0 = \frac{c}{\sigma + \mu} > 1$.

On the other hand, if $G_4(\eta) = \Lambda e^{-6\eta}$, corresponding to the recruitment function

$$g(S, I) = \Lambda e^{-\frac{6I}{(S+I)}},$$

($b=6$), the stability condition (23) is satisfied if

$$6 < \frac{1}{\eta(1-\eta)}, \text{ that is if } 0 \leq \eta \leq \frac{3-\sqrt{3}}{6} \text{ or if } 1 \geq \eta > \frac{3+\sqrt{3}}{6}.$$

Thus the endemic equilibrium is asymptotically stable if $0 \leq \eta < \frac{3-\sqrt{3}}{6}$ or $\frac{3+\sqrt{3}}{6} < \eta \leq 1$ and unstable if $\frac{3-\sqrt{3}}{6} < \eta < \frac{3+\sqrt{3}}{6}$.

A second example is the choice

$$G_4(\eta) = \Lambda e^{-\frac{b\eta}{(1-\eta)}},$$

corresponding to the recruitment function

$$g(S, I) = \Lambda e^{-\frac{bI}{S}}$$

with

$$-G'_4(\eta) = \frac{b\Lambda}{(1-\eta)^2} e^{-\frac{b\eta}{(1-\eta)}} = \frac{bG_4(\eta)}{(1-\eta)^2}.$$

The asymptotic stability condition (23) is $b < \frac{1-\eta}{\eta} = \frac{1}{R_0-1}$. Thus for the model

$$\frac{dS}{dt} = \Lambda e^{-\frac{bI}{S}} - \mu S - c \frac{SI}{S+I} \quad (24)$$

$$\frac{dI}{dt} = c \frac{SI}{S+I} - (\sigma + \mu)I$$

we find that if $R_0 > 1$ there is a unique endemic equilibrium (S_∞, I_∞) given by

$$S_\infty = \frac{\Lambda}{c - \sigma} e^{-b(R_0 - 1)}$$

$$I_\infty = \frac{(c - \mu - \sigma)\Lambda}{(\mu + \sigma)(c - \sigma)} e^{-b(R_0 - 1)}$$

For example, if $b = 1$, this equilibrium is asymptotically stable if and only if $c - \mu - \sigma < \mu + \sigma$, or $c < 2(\mu + \sigma)$. Thus the unique endemic equilibrium is asymptotically stable if $1 < R_0 < 2$ but unstable if $R_0 > 2$. Because the trace of the coefficient matrix passes through zero when $R_0 = 2$ there is a bifurcation to a periodic solution if $R_0 > 2$. Because all solutions of the system (24) remain in the first quadrant and are bounded, the Poincaré – Bendixson theorem guarantees the existence of a stable limit cycle if $R_0 > 2$.

5. SOME EXTENSIONS OF THE MODEL

Generalization of the system (1) is possible in at least two directions.

(a) We may replace the constant per capita death rate μ by a per capita death rate $\mu(N)$ which may depend on total population size. It is then reasonable to assume that $\mu(N)$ is a non-decreasing function of N (only for birth models--included here to facilitate the study of such models),

$$\mu'(N) \geq 0 \quad (25)$$

(b) We may replace the term cSI/N representing the rate of new infections by $\hat{C}(N)SI$. Here $C(N)$ is the number of contacts per infective in unit time and $\hat{C}(N) = C(N)/N$, thus assuming that the

number of contacts per infective in unit time is a function of total population size N . It is then reasonable to assume

$$C(N) \geq 0, \quad C'(N) \geq 0, \quad \hat{C}'(N) \leq 0 \quad (26)$$

for $N > 0$ (Brauer, 1990; Castillo-Chavez, Cooke, Huang and Levin, 1989a, b, c).

We will also impose the requirements

$$\hat{C}(0) < \infty, \quad \hat{C}'(0) < \infty$$

which are not satisfied in (1), where

$$C(N) = c, \quad \hat{C}(N) = c/N.$$

In addition, we define

$$\beta = \hat{C}(K) \quad (27)$$

The model (1) is now replaced by the system

$$\begin{aligned} S' &= g(S, I) - \mu(N)S - \hat{C}(N)SI \\ I' &= \hat{C}(N)SI - [\sigma + \mu(N)] I \end{aligned} \quad (28)$$

We continue to assume that the population has a carrying capacity, and that (4) and (7) are satisfied. We also continue to assume (10) and (11). The equilibrium conditions (13) are replaced by

$$\begin{aligned} g(S, I) &= [\mu(N) + \hat{C}(N)I] S \\ \hat{C}(N)SI &= [\sigma + \mu(N)] I \end{aligned} \quad (29)$$

The linearization of the system (28) at an equilibrium (S_∞, I_∞) , with $N_\infty = S_\infty + I_\infty$ has coefficient matrix [which in the future will be referred as (30)]

$$\begin{bmatrix} g_S(S_\infty, I_\infty) - \mu(N_\infty) - S_\infty \mu'(N_\infty) & g_I(S_\infty, I_\infty) - S_\infty \mu'(N_\infty) \\ -I_\infty \hat{C}(N_\infty) - S_\infty I_\infty \hat{C}'(N_\infty) & -S_\infty \hat{C}(N_\infty) - S_\infty I_\infty \hat{C}'(N_\infty) \\ I_\infty \{\hat{C}(N_\infty) + S_\infty' C(N_\infty)\} - I_\infty \mu'(N_\infty) & -\sigma - \mu(N_\infty) - I_\infty \mu'(N_\infty) \\ & + S_\infty \hat{C}(N_\infty) + S_\infty I_\infty \hat{C}'(N_\infty) \end{bmatrix}.$$

Observe that if $g(0,0) = 0$ it is possible to linearize at the equilibrium $(0, 0)$ provided that $\hat{C}(0)$ and $\hat{C}'(0)$ are finite, which is not true for the choice $C(N) = c$, $\hat{C}(N) = c/N$ used in Section 2. This

analysis is straightforward but is omitted here.

At the disease-free equilibrium $(K, 0)$, the coefficient matrix of the linearization is

$$\begin{bmatrix} g_S(K,0) - \mu(K) - K\mu'(K) & g_I(K,0) - \mu(K) - K\mu'(K) - \beta K \\ 0 & -\sigma - \mu(K) + \beta K \end{bmatrix}.$$

Because of (4), this equilibrium is asymptotically stable if $-\sigma - \mu(K) + \beta K < 0$, or

$$R_0 = \frac{\beta K}{\sigma + \mu(K)} < 1,$$

and is a saddle point if

$$\frac{\beta K}{\sigma + \mu(K)} > 1.$$

The condition for the model (28) to have an endemic equilibrium (S_∞, I_∞) is that the pair of equations (29), or equivalently the pair of equations

$$\begin{aligned} g(S,I) &= N\mu(N) + \sigma I \\ S\hat{C}(N) &= \sigma + \mu(N) \end{aligned} \tag{31}$$

have a solution (S_∞, I_∞) with $0 < S_\infty < K$, $I_\infty > 0$. Because $\sigma + \mu(N)$ is bounded away from zero and $\hat{C}(0)$ is assumed finite (unlike the model (1)), S_∞ is bounded away from zero.

If $R_0 > 1$ the system (28) has an endemic equilibrium. This may be seen by considering the curve

$$g(S,I) = N\mu(N) + \sigma I \tag{32}$$

in the $S-I$ plane running from $(K,0)$ to a point $(0,I_0)$, with $I_0 > 0$ for a recruitment model with $g(0,0) > 0$, $g_I(S,I) \leq 0$. At the point $(0,I_0)$, $\hat{C}(N)S=0$ and $\mu(N)+\sigma=\mu(I_0)+\sigma > 0$. On the other hand at the point $(K,0)$, $\hat{C}(N)S = K\hat{C}(K) = \beta K$ and $\sigma + \mu(N) = \sigma + \mu(K) < \beta K$. Thus there must be a point on the curve (31) at which $\hat{C}(N)S = \sigma + \mu(N)$, and this point is an endemic equilibrium.

There is a unique endemic equilibrium if the directional derivative of the function $\hat{C}(N)S - \mu(N) - \sigma$ along the curve (32) is non-negative, and this is the case provided with

$$[\hat{C}(N) + S\hat{C}'(N) - \mu'(N)] + [S\hat{C}'(N) - \mu'(N)] \left(\frac{g_I(S,I) - N\mu'(N) - \mu(N)}{\sigma + \mu(N) + N\mu'(N) - g_S(S,I)} \right) \geq 0 \tag{33}$$

at all points of the curve (32). Thus if

$$\sigma + \mu(N) + N\mu'(N) - g_S(S,I) > 0 \tag{34}$$

and

$$\begin{aligned} &[\hat{C}(N) + S\hat{C}'(N) - \mu'(N)] + [\sigma + \mu(N) + N\mu'(N) - g_S(S,I)] \\ &+ [S\hat{C}'(N) - \mu'(N)][g_I(S,I) - N\mu'(N) - \mu(N)] > 0 \end{aligned} \tag{35}$$

at all the points of the curve (32), there is a unique endemic equilibrium. In particular, if $g_I(S, I) \leq 0$, the condition (34) is satisfied.

At an endemic equilibrium (S_∞, I_∞) , the matrix (30) has trace

$$g_S(S_\infty, I_\infty) - N_\infty \mu'(N_\infty) - \mu(N_\infty) - I_\infty \hat{C}(N_\infty)$$

and determinant

$$\begin{aligned} & I_\infty [S_\infty \hat{C}'(N_\infty) - \mu'(N_\infty)] g_S(S_\infty, I_\infty) - I_\infty [\hat{C}(N_\infty) + S_\infty \hat{C}'(N_\infty) - \mu'(N_\infty)] g_I(S_\infty, I_\infty) + \\ & \sigma I_\infty [\hat{C}(N_\infty) + S_\infty \hat{C}'(N_\infty)] + I_\infty \mu(N_\infty) \hat{C}(N_\infty) + I_\infty \mu'(N_\infty) [\mu(N_\infty) + I_\infty \hat{C}(N_\infty)]. \end{aligned}$$

The equilibrium is asymptotically stable if and only if the trace is negative and the determinant positive.

If the condition (35) is satisfied at an equilibrium, then it is not difficult to verify that the determinant of the matrix (30) is positive. Further, if both (34) and (35) are satisfied at an endemic equilibrium then the slope of the curve

$$S \hat{C}(N) = \sigma + \mu(N) \quad (36)$$

is greater than the slope of the curve (32) at the equilibrium; recall that an endemic equilibrium is an intersection of the curves (36) and (32).

For a recruitment model, the condition (11) implies that (34) is always satisfied. It is also natural to assume that μ is constant, $\mu'(N) = 0$. At least in the special cases (8) and (9), the condition (35) is satisfied everywhere, showing that there is a unique equilibrium. For (8), with $g(S, I) = G_3(I)$, $G'_3(I) \leq 0$ so that $g(S, I) = 0$, $g_I(S, I) = G'_3(I) \leq 0$, the condition (35) reduces to

$$[\hat{C}(N) + S \hat{C}'(N)] [\sigma + \mu - G'_3(I)] - \mu S \hat{C}'(N) > 0,$$

which is satisfied because of (26), (27), and $G'_3(I) \leq 0$. For (9), with $g(S, I) = G_4\left(\frac{I}{S+I}\right)$, $G'_4(\eta) \leq 0$, so that

$$S g_S(S, I) + I g_I(S, I) = 0, \quad g_I(S, I) = \frac{S}{(S+I)^2} G'_4\left(\frac{I}{S+I}\right) \leq 0,$$

the condition (35) reduces to

$$\begin{aligned} & -I \hat{C}'(N) g_I(S, I) - [\hat{C}(N) + S \hat{C}'(N)] g_I(S, I) + \mu \hat{C}(N) + \sigma [\hat{C}(N) + S \hat{C}'(N)] = \\ & -[\hat{C}(N) + N \hat{C}'(N)] g_I(S, I) + \mu \hat{C}(N) + \sigma [\hat{C}(N) + N \hat{C}'(N)] > 0, \end{aligned}$$

which is satisfied because of (27). If (35) is satisfied the asymptotic stability condition for the endemic equilibrium is (37). Because of (29), at an endemic equilibrium

$$\mu + \hat{C}(N)I = \frac{g(S,I)}{S}.$$

Thus, since $\mu'(N) = 0$, (37) is equivalent to

$$g_S(S,I) < \mu + \hat{C}(N)I = \frac{g(S,I)}{S}.$$

The analysis of this condition for the special cases (8) and (9) is exactly the same as the analysis of the condition (17) for the model (1) in these special cases. We conclude that in the special case (8) the unique endemic equilibrium of the system (28) is always asymptotically stable, while in the special case (9) the endemic equilibrium may be asymptotically stable but may also be unstable with a stable limit cycle oscillation.

In order to describe a non-fatal disease for which a fraction of recovered infectives returns to the susceptible class while the remainder leaves the population, we may replace the model (28) by

$$S' = g(S,I) - \mu(N)S - \hat{C}(N)SI + aI$$

$$I' = \hat{C}(N)SI - [\sigma + \mu(N)] I$$

with $0 \leq a \leq \sigma$. The matrix (30) would then contain an additional term a in the first row, second column. This would have no effect on the trace of the matrix and would subtract a $I_\infty[\hat{C}(N_\infty) + S_\infty \hat{C}'(N_\infty)]$ from the determinant. If $0 \leq a \leq \sigma$ this would not change the sign of the determinant or the validity of the condition (35). Thus the inclusion of this internal recruitment term, which may be appropriate in modelling diseases such as gonorrhea, would not affect the qualitative behavior of the system, except for possible effects of moving the equilibrium and thus changing equilibrium values of the entries of the matrix (30). It would, however, tend to lengthen the period for damped and undamped oscillations. Some simulations suggest that the effect of increasing a is to decrease the amplitude of a limit cycle and thus to give the appearance of stabilizing the endemic equilibrium. These simulations were carried out for the model

$$\frac{dS}{dt} = \Lambda e^{-\frac{bI}{(S+I)}} - \mu S - c \frac{SI}{S+I} + a I$$

$$\frac{dI}{dt} = c \frac{SI}{S+I} - (\sigma + \mu) I$$

using parameter values (time measure in days) $\Lambda=0.01$, $\mu=0.001$, $\sigma=0.03$, $c=0.05$, appropriate for gonorrhea (see Hethcote and Yorke 1984), and $b=6$. If $a = 0$ there is a limit cycle over which S varies between 0.1 and 1.12 while I varies between 0 and 0.25. If $a = 0.003$ S varies between 0.1 and 0.7 and I varies between 0.02 and 0.2. If $a = 0.01$ S varies between 0.08 and 0.12 and I varies between 0.03

and 0.06. These ranges should be compared with the carrying capacity 10 of the core group.

6. CONCLUSIONS

A central problem associated with the study of the transmission dynamics of STD's is that of determining what is the population at risk. The careful analysis of this question brings us back to the fact that in order to understand STD dynamics one must define clearly the interacting populations, especially their sizes and the nature of their interactions (who is mixing with whom). Theoreticians and mathematical epidemiologists in the past have avoided this question by studying disease dynamics in the context of closed populations. Furthermore, it has been customary to concentrate on the study of disease dynamics under the assumption of the existence of subpopulations with a fixed number of individuals. Consequently, the effectiveness of disease management measures can only be evaluated through the quantitative magnitude of the shifts of individuals between the susceptible class and the infective class (in the case of gonorrhea, see Hethcote and Yorke 1984). Classical studies concentrate on the potential effects that a very sexually active group of individuals--the core group--has on disease dynamics within a closed social network involving population nodes with a fixed number of individuals (but see Castillo-Chavez *et al.* 1992, Rubin *et al.* 1991).

The core group as well as all interacting subgroups are assumed to have a fixed number of individuals and changes in behavior are measured by looking at changes in the endemic equilibrium which is a function of parameters which may be sensitive to management. In this manuscript, we have followed a different approach. We have ignored the explicit dynamics of all groups except for that of the core group, yet do not assume that it lives in the vacuum or in an indifferent world. The core may gain and/or lose individuals from the population at large. Some of these gains or losses may be temporary while others may be permanent. Studying the dynamics of the core population under this assumption may produce substantial changes in the total number of individuals belonging to the core. Hence, we need to study the disease dynamics of the core group in the context of mathematical models with variable population size.

Our results show that this view--from the core--does indeed offer new possibilities and new metaphors that need to be considered in the study of STD dynamics. Our models suggest that changes in behavior may produce substantial quantitative and qualitative changes in disease dynamics. The size of the core population at equilibrium may experience a considerable reduction in reaction to a recruitment function that depends very heavily on changes in behavior, even to the point of changing the qualitative dynamics of the epidemic among the core (sustained oscillations).

There are several possible extensions of this work. The response of the meta-population to the disease prevalence is not usually immediate. One can easily envision several realistic situations in

which this is indeed not the case. We plan to study model (1) when a delayed response in recruitment is observed. In general the HIV/AIDS epidemic is not well modelled by (1). We may need to change the distribution of the infectious periods from negative exponential distribution to a generalized gamma distribution. Furthermore, in this situation g may be given as $g = g(A)$ where A denotes those individuals with “full-blown” AIDS. That is, the meta-population may be reacting to the number of AIDS cases rather than to the unknown number or proportion of HIV positive.

The potential theoretical use of the results of this manuscript may be better understood in the context of H. V. Fineberg’s 1988 remark: “Perhaps, as more people are touched personally by AIDS, our collective resolve to stem the epidemic will be fortified. These considerations point to a sad, and somewhat paradoxical, impression; our country may have to experience more spread of infection in order to prevent spread.”

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